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Dr. James Ewing, *President.*

OBSERVATIONS ON MALARIAL PARASITES.

L. B. GOLDHORN, M.D.

In protracted cases of tertian fever, certain forms of the parasite are quite constantly met with, which differ essentially from the ordinary ones of the asexual cycle. They take no part in segmentation, and they are never seen in the recent primary infections. At least two of them are the analogue of the crescentic and ovoid bodies of tropical malaria; they are the macrogamete and the microgametocyte of tertian fever. They are readily recognized when fully developed, in the following manner: in smears treated with chromatin dyes, the male or microgametocyte is a rather large parasite with large achromatic zone, in which a mass of unevenly staining chromatin lies in the form of filaments. The parasite's body takes the basic dyes poorly and its coarse, blackish pigment is brought out more distinctly than in any other

form. It never has multiple chromatin bodies. In the moist chamber it flagellates and the flagella originate within a limited area which corresponds to the zona achromatica. Before it flagellates, its usually quiescent pigment beomes unusually agitated. It disengages itself from the corpuscular remnant, assumes a spherical form, and flagellates. In stained preparations many variations in the position of the chromatin mass are seen, but on the whole the characteristics remain those given. The female or macrogamete is still larger when fully developed; in the chromatin stained smear it presents a large well stained body with or without corpuscular remnant and a small irregular chromatin mass in a poorly defined zone situated rather constantly peripherally, rarely ever centrally, but at times just within the body. Its pigment is somewhat finer than that of the microgametocyte. A third form has a round chromatin kernel well within the parasite's body. This kernel is surrounded by a lightly stained reticulated area which also takes on the chromatin dye and is therefore pink. The body of this form stains well with methylene blue and it is usually smaller than the two forms just described. I have never seen it extra corpuscularly, and believe it to represent no phase of the schizogony, as I have never met with it in recent primary cases. Much confusion arises from the promiscuous study of parasites in cases of relapse, or old cases. Many typical forms are seen, such as multiple chromatin bodies in young forms, premature segmentation, abnormal position of chromatin, and queer positions of the parasites on the corpuscle. But the number of segments (schizonts) varies even in perfectly fresh cases. As each young parasite receives an equal share of the mother's body and as there is no waste apart from the transformed haemoglobin and corpuscular

remnant, it seems to me that the number of segments depends upon the size of the infected corpuscle and upon the amount of nourishment it contains. (Double infections of one corpuscle might also be considered, but frequently one parasite atrophies). As the parasite forming the greatest number of segments might require more time for the completion of the cycle than one forming a small number only, it would by segmenting later fall behind the other parasites. This irregularity, if exaggerated, might influence the length and periodicity of the paroxysms. As a matter of fact one can find segmenting bodies for some time after a chill, and sometimes the duration of the chill is protracted in advanced cases. By diligent study of animal malarial parasites I have found much which has led to a better understanding of human malaria. In a paper read before the University Medical Society I described a parasite found in a species of African monkey. I have since had occasion to study the parasite again and now feel certain that nearly all forms seen by me were gametocytes. What I stated about the staining and chromatin characteristics of the human tertian parasite is, in the monkey, still more clearly defined; and the third form with karyosome and paler staining reticulated area is readily found. Febrigenous forms were not seen. Bone marrow and spleen contained no segmenting bodies. A peculiar notch at the edge of this last (third) form and of the macrogamete was rather constantly observed and helped to confirm the belief that these third forms are related to the gametes, and most closely to the macrogametes. The parasite obtained from the monkey is probably the analogue of tertian fever. In birds with Halteridium infection one sees the above enumerated characteristics of gametocytes. The staining differences

and those in the chromatin are pronounced. The microgametocyte stains very poorly, has a very large central achromatic zone and more or less polar pigment; in the achromatic zone lie delicate filaments of chromatin. The deeply staining macrogamete has, on the other hand, a small chromatin mass usually at the edge in a small achromatic zone, and the relatively finer pigment lies all over the body of the protozoon. I believe these "hantelforms" to be the analogue of the human estivo-autumnal gametocytes, the so-called crescents. Several preparations in my possession show some details in crescentic bodies which I have not seen demonstrated before. If the pigment be removed from these gametocytes a large achromatic zone becomes wonderfully distinct; in it lies the chromatin always in a single body, never split up into segments. As a matter of fact, I have never seen evidence of parthogenesis in crescents. There are some curious differences in the staining of crescents; some have marked polar staining, others stain lighter blue throughout. It is possible that the one staining deeper may be the female. This year it was impossible to obtain cases with crescentic bodies and experimental work that had been planned had to be abandoned. Most instructive material was, however, obtained in the form of tertian cases. Only a brief review of the more interesting cases may be given. An old case of one and a half year's standing with frequent relapses showed only a few queer young forms and many gametocytes; no parasites with multiple chromatin bodies were found. Another case of seven weeks' standing with rich infection showed an unsual number of corpuscles with two parasites maturing side by side, or one developing at the expense of the other. A case of a woman from Coste Rica, where the malaria was contracted, was most instructive

because no strictly normal segmenting bodies such as one is accustomed to find in the recent primary infection were seen; instead there were ring forms with small accessory chromatin body in the thickened portion of the body, or two, three or even five unevenly staining, large round chromatin bodies in a large ring of even diameter throughout. There were never more than twelve segments, usually not over ten; and the parasite's body was always exceptionally small. Yet the case was one of tertian fever. A case with indistinct malarial history in a child a little under ten years showed large ring-like parasites with a single chromatin body and large achromatic zone with heavy pigment, in a deeply notched corpuscle, but totally unlike the rings seen in estivo autumnal cases in the crenated corpuscles. Many more instances might be cited. It seems to me that malarial parasites form what one might term involution forms at a time when gametocytes (which I consider an expression of the necessity of sexual reproduction) make their appearance. The parthoginetic impulse exhausts itself and the vitality of the febrigenous forms is lowered. One cannot know how much the production of possible antibodies may have to do with the phenomenon. That such bodies are formed is reasonable to suppose, as cases of immunity exist, and the study of kindred hematozoa (of trypanosoma, for instance) helps to confirm this belief. To-day, the practioner who owns a microscope is as a rule satisfied with the finding of the hematazoon of tertian fever, but the kind and form and characteristics are not usually considered. As soon as all typical forms have been recognized a better understanding of the asexual cycle and gametocyte formation will be reached. If gametocyte formation could be prevented, transmission of malaria from one individual to another by mosquitos

might also be prevented, as the perpetuation of the parasite depends upon them; hence, cases containg the more resistant gametocytes would require greater attention and possibly somewhat different treatment than cases of recent infection with immature parasites. The value of positive recognition of mature forms (gametocytes) in tertian fever, therefore, becomes paramount.

Discussion.

Dr. Ewing: I have been extremely interested in the facts brought out by Dr. Goldhorn. I believe that the question of the differentiation of the sexes in malaria is at the present time one of live interest. I have been over much of this ground without, however, getting all the points obtained by Dr. Goldhorn. Some two years ago I thought that I was able to identify the macro- and microgametes, but was not certain because I was not able to follow the cases through a series of paroxysms. Since that time one writer at least has attempted to differentiate the male and female parasites in the blood. Argutinsky, (*Arch. f. Micr. Anat.*, Bd. 59, 1902.) As the malarial paroxysm progresses the morphology changes and the male and female parasites become clearly differentiated. I do not agree in every particular with Dr. Goldhorn's conclusions. In the first place, Dr. Goldhorn found that the male stained lightly with methylene blue; in my specimens the male stained darker than the female, though that was possibly due to some difference in technique. I have always found, as did Dr. Goldhorn, that the flagella are extruded from one point in the microgamete, but before the extrusion of flagella I have sometimes seen the chromatin subdivided into four or six groups.

SEVERAL CASES OF DYSENTERY REFERABLE TO THE BACILLUS OF SHIGA.

W. H. PARK, M.D.

I recently became interested in dysentery while staying at Mt. Desert this summer with Dr. E. K. Dunham and Dr. Flexner. There was an outbreak of a number of cases of dysentery in some of the families living nearby. There were probably twenty cases in all, occurring mostly in children. Dr. Flexner kindly sent to Philadelphia for cultures of the Shiga bacillus so that in case of failure to obtain cultures from these cases they might test the serum reaction. The day the cultures came, Dr. Dunham succeeded in getting serum from two children sick with dysentery and both specimens gave a marked reaction with the cultures from Philadelphia. The same day I saw a case of dysentery and obtained some mucus directly after its expulsion from the rectum. This was plated, and I got the dysentery bacillus of Shiga, which I now show you in the tubes. This bacillus appears to have the same characteristics as those isolated by Shiga in Japan and by Flexner in this country and in the Philipines. I also show you three tubes showing the growth in the Hiss media of the Shiga dysentery bacillus, and of the colon and typhoid bacilli. The dysentery bacillus remains isolated and does not disseminate through the media nor produce gas. The colon bacillus passes throughout the media producing a general cloudness varied by little streaks due to the passage through the media of gas bubbles. The typhoid also passes throughout the media but causes no appreciable gas and therefore leaves no streaks. This investigation made therefore the interesting fact apparent that there was an epidemic in Mt. Desert in

which the Shiga bacillus was the exciting factor giving rise to acute dysentery. On my return to the city, I thought it interesting to see how much dysentery was present in New York and how prevalent was the Shiga bacillus. Inquiries made among the hospitals and asylums developed the fact that there was very little dysentery then present in the city. On case, accidentally found, was that of a man who had come from the Philipines three years before, and who ever since had had subacute dysentery. For two or three weeks he would be fairly well, never entirely so, and then he would have an acute attack. His blood showed a marked reaction with the Shiga bacillus, and this bacillus, as well as the amœba coli was found in his stools. This case then seems to be a case a chronic dysentery due to mixed infection with the amœba coli and the Shiga bacillus. Two cases were found, of a man and a baby, who had had dysentery six weeks before and whose blood gave the specific reaction. In thirty cases where the blood from suspected typhoid patients was examined one gave a reaction with the Shiga bacillus. This blood caused agglutination of both the typhoid and dysentery bacilli. The blood of a number of children who had recently suffered from acute entero-colitis did not agglutinate the dysentery bacilli even when used in a dilution of one part of serum to ten of bouillon. This is interesting in view of the preliminary report from Baltimore where the Shiga bacillus was abundantly present in the stools of children suffering from acute entero-colitis.

A SOLITARY TUBERCLE OF THE BRAIN.

DAVID BOVAIRD, M.D.

The specimen which I present is of rather peculiar clinical interest. It was taken from a girl six years of age, whose family history was entirely negative. When first seen, November 29, she gave a history of having had a fall the previous July. The symptoms dated from that time. Two weeks later there was a loss of power in the right leg, and still later in the right arm and right side of the face. On examination there was found to be a paralysis of most of the muscles of the leg and of the muscles of the arm as well. The tongue deviated to the right on protrusion and on smilling the right side of the face showed no action. A diagnosis was made of cerebral paralysis. A brace was applied to prevent the dropping of the leg, but the improvement was not satisfactory and in March she was admitted to the hospital for the correction of the condition in her right leg. Upon her admission her leg was put in plaster of Paris. Finally the child developed symptoms of pneumonia and later gradually developed meningitis. The disease ran the ordinary course of tuberculous meningitis and the child died. At autopsy, the brain was found generally congested. Over the base of the brain there was a thick greenish-yellow exudate. In the sulci, especially along the middle cerebral arteries were multitudes of miliary tubercles. In the left hemisphere, just beneath the upper end of the fissure of Rolando and about one cm. from the surface, was the solitary tubercle, about four cm. in diameter presented as a specimen. Immediately posterior to this was a second mass, about one-half the diameter of the first. Several bronchial glands were tuberculous; one was calcareous. There were scattered areas of broncho-

pneumonia in the lungs. The viscera was otherwise normal. This case was of especial interest because of the size of the tubercle, which was very unusual for a child of six years—the largest that I have ever seen in a child's brain. The history is interesting as evidence that this solitary tubercle developed as a tumor of the brain and was responsible for the condition found in the right leg, in the right arm, and in the right side of the face, and that the tuberculous meningitis was a secondary development following the child's admission to the hospital. It is the only case of this character that I have ever come in contact with—a solitary tubercle developing in the brain of a child just as a tumor might have done, and during its early stages entirely devoid of any symptoms pointing to its tuberculous nature.

A CASE OF TRAUMATIC DISEASE OF THE AORTIC VALVE.

E. LIBMAN, M.D.

The heart which I present was obtained at the post-mortem examination of a man thirty-three years of age, who had succumbed to an infection by a paracolon bacillus. The history of the case in so far as it appertains to the heart was as follows:—There was hypertrophy and some dilatation of the left ventricle. There was a slight systolic murmur at the apex and over the aortic valve. There was a rough systolic murmur over the pulmonic orifice, without visible pulsation. The second sound was louder over the pulmonic than over the aortic orifice. The murmur was not heard in the vessels of the neck. The diagnosis wavered between several conditions. In the first place, the patient had had six children, nearly

all of whom had died in early infancy, having been "blue babies". This being taken into consideration, it was thought possible that the patient might have a similar condition, but no one of the usual congenital lesions in itself would very well account for the physical signs present. The possibility of an acute endocarditis due to the paracolon bacillus was also entertained. A lesion of the aortic valve in which the murmur was heard over the pulmonary orifice was the third diagnosis considered. The relatives of the patient stated that he had been perfectly well up to the age of three years, when he was run over and three of the upper ribs on the left side anteriorly, were fractured. After that, a very marked cardiac impulse had been noticed and he suffered from palpitation. It could not be ascertained whether or not fever was present after the injury. The lesion found in the heart is of such a nature that it can only be explained as the result of the traumatism. The anterior and the right posterior flaps of the aortic valve were found torn loose from their attachment to the aorta. There was considerable thickening found at the place from which the flaps were detached, and the two flaps were found to be agglutinated by fibrous tissue with calcareous deposit. Furthermore, there was a line of calcareous tissue running directly across the anterior flap, which also extended down over the endocardium for a distance of about one-half of a centimetre. There was no atheroma of the aorta. One cannot help coming to the conclusion that the trauma sustained thirty years before death had resulted in a tear across the anterior flap of the valve and in a detachment of two flaps from their attachment to the aorta. The question remains open whether the changes found were due in part to an infectious process following the traumatic lesions or not. Prof. Prudden

has shown that endocardial changes may result after trauma without the occurrence of any infection.

MIXED TUMORS OF THE PAROTID.

F. C. WOOD, M.D.

Two mixed tumors of the parotid have recently come under my observation. In one of these tumors, which showed a typical endothelial type of growth, there were numerous epithelial pearls containing spine cells. The second case is a tumor of long duration in which flat celled carcinoma developed in the substance of a congenital endothelial tumor.

Discussion.

DR. LIBMAN: During the past year we have had at the Mt. Sinai Hospital laboratory a case of mixed tumor of the parotid in which we found distinct areas of epithelioma.

A CASE OF GENERAL TUBERCULOSIS.

G. ROPER, M.D.

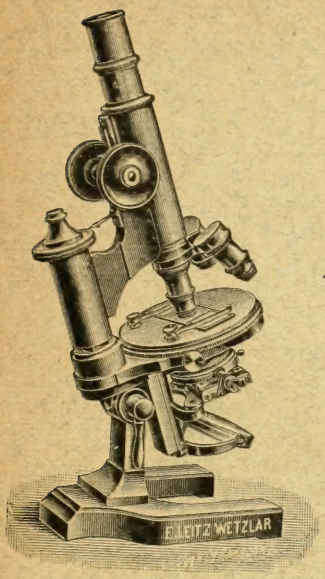
I present this specimen because of the condition observed in one of the ovaries. The patient was a colored woman, aged thirty-seven years. Seven months previously, after giving birth to her fourth child, she had had a slight cough and expectoration. Two weeks after delivery, night sweats began and she lost flesh and had considerable pain in the abdomen. On physical examination there was found to be a distended mass in the abdo-

men with considerable fluid. At autopsy the retro-peritonial glands were found to be enormously enlarged and tuberculous. There was also a general miliary tuberculosis of the spleen and lungs. The right ovary was filled with numerous large tubercles. The other ovary was normal. The exact seat of the original lesion could not be definitely determined,

Discussion.

DR. EWING: Were any intestinal lesions found?

DR. ROPER: There were no intestinal lesions. Dr. Biggs had been inclined to think that the pulmonary glands might have been the original seat of the trouble; but they were but moderately involved, while the retro-peritoneal glands were enormously enlarged.



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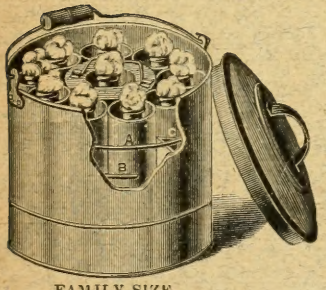

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